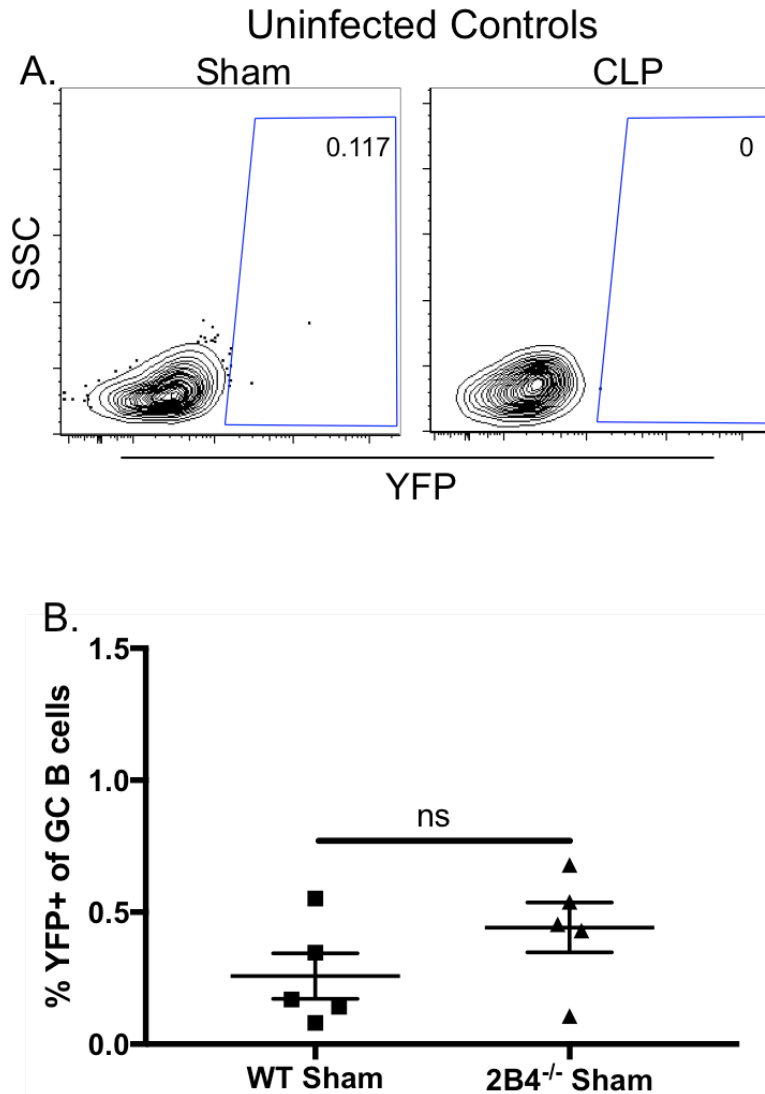
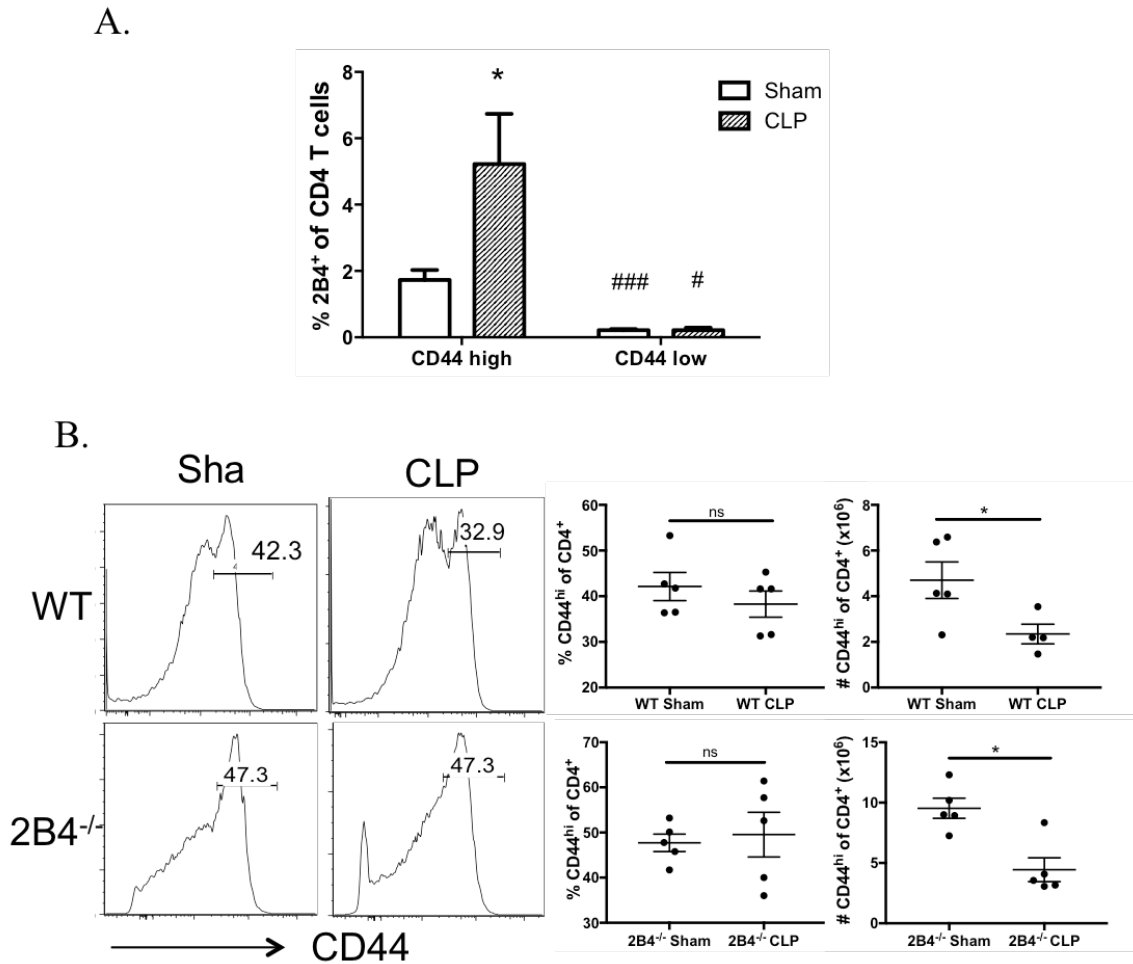


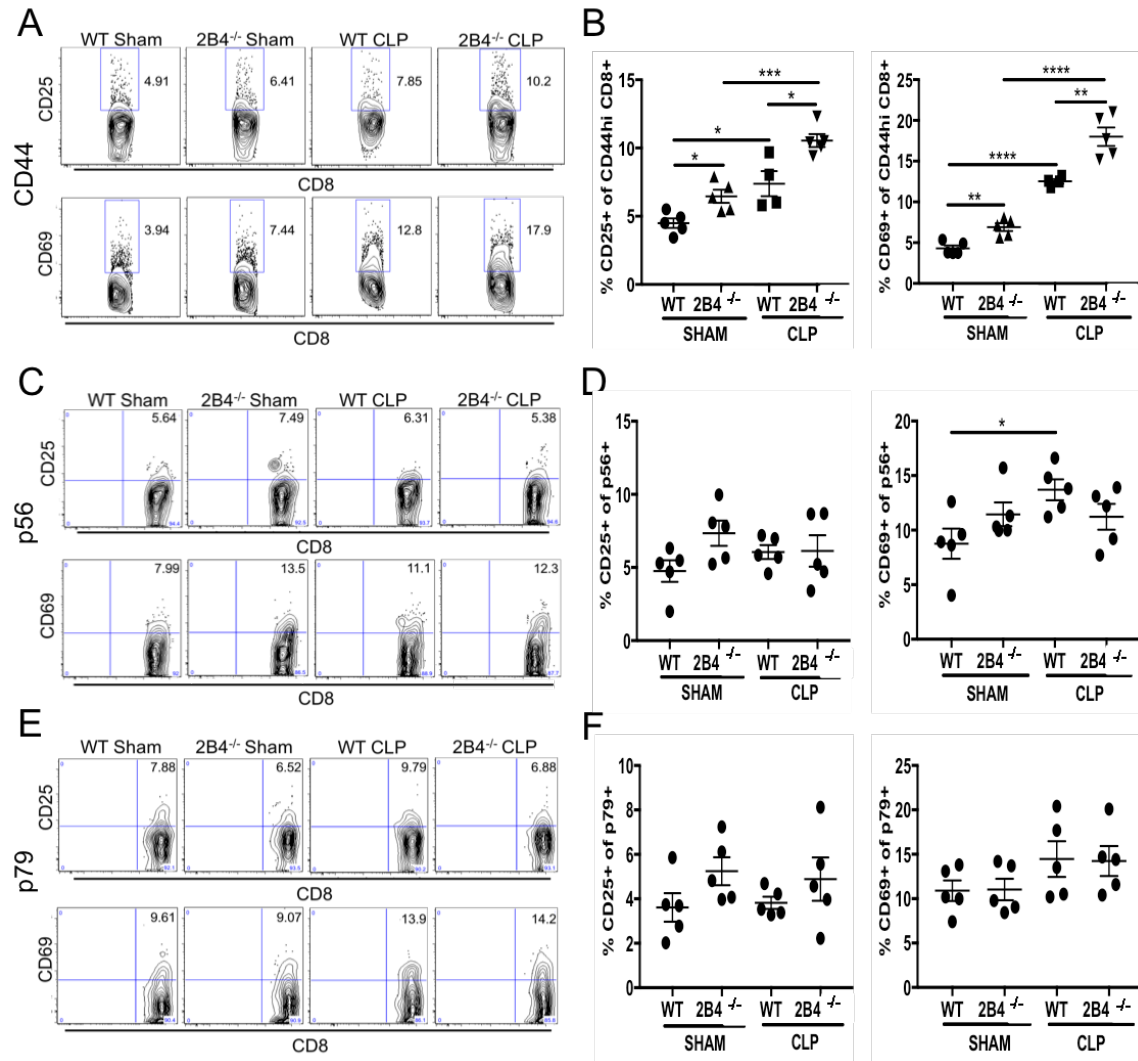
Supplemental Figure Legends



Supplemental Figure 1. gHV viral load is not different between WT and 2B4^{-/-} sham animals. (A) Negative controls showing background staining of YFP in uninfected animals. Uninfected age-matched animals were subjected to CLP or sham surgery and sacrificed 3 days later (33 days post mock infection). Splenocytes were stained with the same flow cytometry panels used for infected experimental animals, and level of background staining in the YFP channel was assessed. (B) WT and 2B4^{-/-} animals were infected with gHV and subjected to sham surgery 30 days later. Viral burden was assessed via analysis of YFP expression in GC B cells in the spleen 3 days later. Results show there is no difference in the viral burden between WT and 2B4^{-/-} animals in the absence of CLP. Data are representative of 2-3 independent experiments with 5 mice per group. Statistical analysis was conducted using a Student's t-test.



Supplemental Figure 2. 2B4 is upregulated within the CD44^{hi} CD4⁺ T cell subset following CLP. Three days post CLP and 33 days post gHV infection, splenocytes from WT sham or CLP animals were assessed for the proportion of CD4⁺ T cells expressing the coinhibitory molecule 2B4. (A) 2B4 expression on CD4⁺ T cells taken from sham and CLP animals. Increased proportions of CD44^{hi} T cells expressed 2B4 compared to CD44^{lo} T cells following CLP (A). (B) Naïve WT and 2B4^{-/-} mice were infected with 2×10^3 PFU intraperitoneally of a recombinant, transgenic murine gammaherpesvirus (gHV), MHV68-H2^b-YFP. Data shown are representative flow plots and summary data of percent (left) and absolute number (right) of CD44^{hi} CD4⁺ T cell populations in WT sham and CLP animals (top panels) and 2B4^{-/-} CLP animals and 2B4^{-/-} sham controls (bottom panels). Data are representative of 2-3 independent experiments with 5 mice per group. Statistical analysis was conducted using a Student's t-test. *p<0.05.



Supplemental Figure 3. 2B4 deficiency did not improve antigen-specific CD8⁺ T cell activation following sepsis. Three days post CLP and 33 days post gHV infection, splenocytes from WT or 2B4^{-/-} sham or CLP animals were assessed for the proportion of cells expressing the early activation markers CD25 and CD69. Representative flow plots of CD25 (top) and CD69 (bottom) of CD8⁺ CD44^{hi} antigen-experienced T cells (A), as well as gHV-specific p56⁺ (C) and p79⁺ (E) populations. The data is summarized in (B, D, and F, respectively). Data are representative of 2-3 independent experiments with 5 mice per group. Statistical analysis was conducted using a Student's t-test. *p<0.05, **p<0.01, ***p<0.001, ****p<0.0001.